intravenous infusion of ginsenoside Rb_1 starting after cerebrovascular occlusion exhibited a marked favorable effect on rats with permanent MCA occlusion clearly indicates the usefulness, convenience and economical advantage of intravenous infusion of ginsenoside Rb_1 in low doses.

On the other hand, in the previous report by the inventors of the present invention (Sakanaka and Tanaka), in which ginsenoside Rb_1 was directly infused into the cerebral ventricles of animals with MCA permanent occlusion (Zhang B., et al., J. Stroke Cerebrovasc. Dis., 7, 1-9, 1998), a significant suppressive effect on cerebral infarction was observed only when the continuous intracerebroventricular infusion of ginsenoside Rb_1 at the dose of 0.6μg/day was conducted after MCA occlusion; and the effect was equal to or a little less than the effect of intravenous administration of ginsenoside Rb_1 as shown in the present example. In the previous report on the intracerebroventricular administration of ginsenoside Rb_1, no therapeutic effect on cerebral infarction was observed when the other doses of ginsenoside Rb_1 (6μg/day or 0.06μg/day) were continuously infused into the cerebroventricles after MCA permanent occlusion. Consequently, the effective dose range of intracerebroventricularly administered ginsenoside Rb_1 was very narrow and its practical use for clinical medicine was thought to be difficult. Moreover, the actual application of intracerebroventricularly infused ginsenoside Rb_1 to humans

appears to be impossible when we consider the balance between its risk and benefit.

Generally, a neuroprotective factor or agent exhibits the maximum effect when directly administered in the cerebral ventricles or into the brain parenchyma, and in case of intravenous or intraperitoneal administration, its effect and efficacy seem to drastically decrease or disappear due to the blood brain barrier that prevents the neuroprotective agent from entering the brain parenchyma, or due to metabolic decomposition of the agent. Consequently, based on the experimental results of intraperitoneal administration or intracerebroventricular administration of ginsenoside Rb_1, the effect and efficacy of intravenously infused ginsenoside Rb_1 could not be anticipated at all.

As clarified by the present invention, however, intravenous administration of ginsenoside Rb_1 reduces effectively the cerebral infarct area of rats with MCA permanent occlusion in a wider dose range than in case of intracerebroventricular administration, and improves learning ability of the MCA-occluded animals. Ginsenoside Rb_1 is a purified saponin, which is contained in medicinal ginseng, but since it can not be detected in blood after oral administration, a pharmacological action of ginsenoside Rb_1 per se has been substantially denied (Kobashi et al., Medicinal ginseng '95, pp 213-221, Ed. Kumagai A., Kyoritsu Publ.). However, according

to the present example, as described in JP98/365560 and PCT/JP99/02550 ("Brain cell or nerve cell-protective agents comprising ginsenoside Rb_1"), it is cleared that intravenous administration of ginsenoside Rb_1 has effect, efficacy and use independent of the medicinal ginseng.

Next, we prepared paraffin sections, 5 μm thickness, obtained from brain samples at the level about 2.8 mm posterior to the bregma. Then, we measured the cerebrovascular area per 1.27 mm^2 of the non-infarcted ischemic penumbra in the parietal lobe. This was done for each of the cerebral hemisphere of a group to which ginsenoside Rb_1 had been administered intravenously (i.e. the brain tissues had been rescued by administration of ginsenoside Rb_1) (Fig. 5). Four sheets of differential interference contrast micrograph were used for measurement (Fig. 6), and the ratio of the cerebrovascular area was calculated (Table 1).

Table 1

	intact side (%)	ischemic side (%)
Rb_1: 6μg/day	7.0 ± 0.64	8.0 ± 0.58
Rb_1: 60μg/day	8.3 ± 0.92	9.0 ± 0.52

The ratio of the cerebrovascular area was also measured